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Italian Article

ACUTE MYOCARDIC ISCHEMIA AND THYROTOXICOSIS: RAPID REGRESSION OF ISCHEMIA WITH PROPRANOLOL AND PROPYLTHIOURACIL - A case report

[Ischemia miocardica acuta in corso di tireotossicosi: regressione rapida dell'ischemia con l'impiego di propranololo e propiltiouracile. Descrizione di un caso clinico]

C. Della Corte, R. Della Corte and M. Festa

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ACUTE MYOCARDIC ISCHEMIA AND THYROTOXICOSIS: RAPID REGRESSION OF ISCHEMIA WITH PROPRANOLOL AND PROPYLTHIOURACIL

- A case report

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(Primary: Dr. C. Della Corte)

Acute ischemic heart disease and thyrotoxicosis: rapid regression of myocardial ischemia with propranolol and propylthiouracil. A case report.

The authors report a case of a female patient who during thyrotoxicosis developed acute ischemic heart disease that remained unchanged by therapy with transdermal nitroglycerin, calcium-channel blockers and heparin calcium, but promptly healed with propranolol and propylthiouracil. The authors remark the importance of early therapy with beta blockers in acute ischemic heart disease associated with thyrotoxicosis.

Key words: Ischemic heart disease - Thyrotoxicosis - Propranolol.

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* Student in Medicine

The cardiac manifestations of thyrotoxicosis are in general represented by disorders of rhythm, in particular from atrial fibrillation.

Nevertheless, at times thyrotoxicosis may be associated with chronic ischemic cardiopathy, or it may be the source of acute myocardial ischemia[1].

We thought it interesting to describe a case that we recently observed in the Hospital of Orte. It was a patient affected with hyperthyroidism, who during a thyrotoxic crisis developed acute myocardial ischemia that was not influenced by nitroderivatives and by calcioantagonists, but promptly regressed with the use of a beta blocker (Propranolol), which administered together with Propylthiouracil, also brought about the resolution of the thyrotoxic crisis.

Clinical Case

B.M.G. Patient of female sex, 67 years of age, married, with 8 children.

Nothing of significance in the physiological anamnesis.

The remote pathologic anamnesis evidenced cholelithiasis from 1982 and hyperthyroidism from 1983 (T3 = 250 ng/dL, T4 = 14 µg/dL, TSH = 0.1 µU/mL).

In 1987 the patient was subjected to therapy with Methimazole, 1 tablet 3 times a day, which brought about the appearance of petechiae after several months of treatment. For this reason, the drug was suspended and never again resumed. Since then, the patient neither followed another "thyrostatic" therapy, nor controlled thyroid function. Out-patient ECGs always turned out normal.

Twenty days before entering our hospital, the patient had presented precordial pain irradiated to the back, palpitations, frequent episodes of diarrhea, mild muscular tremors, insomnia, irritability.

A ECG had demonstrated atrial fibrillation and high ventricular frequency (160/m'), but not significant alterations of tract ST-T.

The patient had been treated at home from the start with Desacetyl Lanatoside C and Dihydroquinidine and then with Digoxin and Verapamil, which had brought about regression of the atrial fibrillation with renewal of sinusal rhythm.

The hematochemical examinations done at home evidenced normality of CPK, CPKMB, transaminases, LDH, glycemia, azotemia, creatinine, Na, K, Ca, hemochrome, platelets, urine examination,

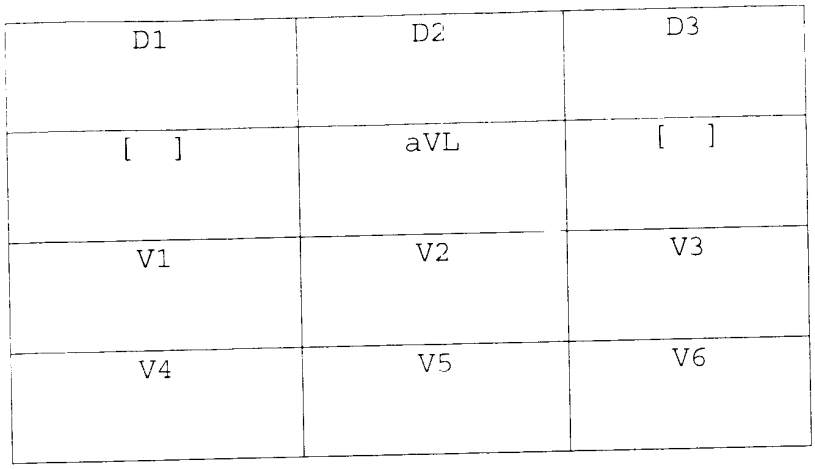

Fig. 1 - ECG of B.M.G. upon entering the Hospital of Orte. Sinusal rhythm. Cardiac frequency 72/m'. PR = 0.18'. Horizontal electrical position and left axial deviation. ST tract slightly marked and T wave clearly negative in D1, D2, aVL, V2, V3, V4, V5, V6.

prothrombin time, PTT, while T3 was 400 ng/dL, T4 18 µg/dL, and TSH 0.06 µU/mL.

Successive home ECGs evidenced sinusal rhythm and T wave inversion in D1, D2, aVL, V2, V3, V4, V5, V6, as if from acute myocardial ischemia. For this reason, to the therapy with Digoxin and Verapamil were added nitroderivatives (Nitroglycerin by transdermal administration, 10 mg, 1 patch a day) and Heparin Calcium (5,000 U subcutaneously, 3 times a day), without result with regard to the ischemia. Consequently, the patient was

recovering in our Hospital.

The objective examination upon admission evidenced bilateral exophthalmus, anxious facies, palpable thyroid, ample and shooting pulse, rhythmic cardiac activity, positivity of symptoms of Stellwag, Graefe, Moebius and Joffroy, liveliness of the osteotendinous reflexes, mild hand tremors.

The patient also presented frequent diarrhoic discharges. Arterial pressure was 150/70 mm Hg, the thorax X-ray and hematochemical examinations on admission proved normal, except T3 = 400 ng/dL, T4 = 20 µg/dL, TSH = 0.04 µU/mL, cholesterol = 135 mg%.

The search for cardiotropic antivirus antibodies proved negative.

The thyroidal echography permitted discerning a thyroid enlarged in total volume, with dishomogenous echostructure due to the presence of multiple nodule formations.

The ECG on admission (Fig. 1) confirmed T wave inversion in D1, D2, aVL, V2, V3, V4, V5, V6.

On the basis of these data, a diagnosis of acute myocardial ischemia with thyrotoxicosis was postulated, and the patient was treated with Propranolol 40 mg, 1 tablet 2 times a day, and Propylthiouracil 50 mg, 3 times a day by mouth.

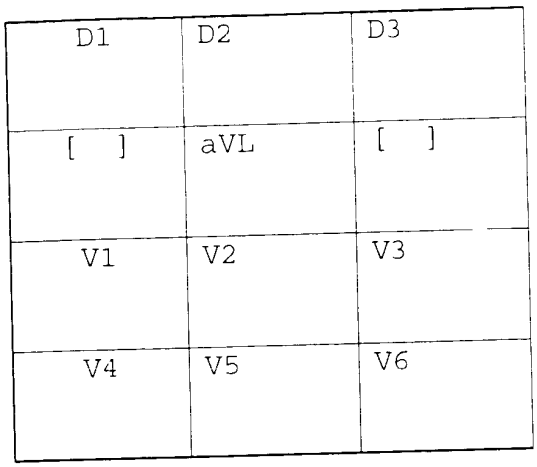

Fig. 2 - ECG of B.M.G. after 5 days of therapy with Propranolol and Propylthiouracil. Sinusal rhythm. Cardiac frequency 56/m'. PR = 0.18'. Horizontal electrical position and left axial deviation. Isodiphasic T wave in D1, aVL, V2, V3, V4, V5, V6.

After 5 days of such therapy, the ECG (Fig. 2) showed a sharp improvement of the ischemic symptomatic picture, with isodiphasic T wave in D1, aVL, V2, V3, V4, V5, V6.

After another 4 days of therapy, the ECG proved practically normal (Fig.3). Echocardiography also showed a symptomatic picture within normal limits.

A successive control series of ECGs confirmed the complete regression of myocardial ischemia, while after 3 months of therapy T3 was 200 ng/dL, T4 12 µg/dL, and TSH 0.8 µU/mL.

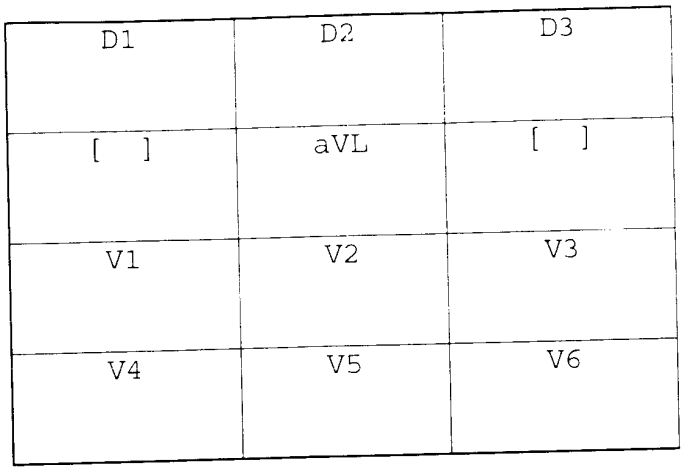

Fig. 3 - ECG of B.M.G. after 9 days of therapy with Propranolol and Propylthiouracil. Sinusal rhythm. Cardiac frequency 60/m'. PR = 0.18'. Horizontal electrical position and left axial deviation. ECG practically within the norm.

With this therapy was likewise observed rapid regression of the clinical symptoms of thyrotoxicosis.

The patient refused to perform myocardial scintigraphy and coronarography.

Discussion

Alterations of cardiac rhythm, in particular atrial fibrillation and extrasystolia, are the most classic manifestations of thyrotoxicosis.

Hyperthyroidism can also be the cause of myocardial ischemia with discrepancy between hemodynamic possibilities of the coronaries and oxygenative requests of the myocardium, enhanced by the increased adrenergic tone which it comports (ischemic cardiopathy of integral coronaries with hyperthyroidism)[1,4]. More often, however, it happens that a latent ischemic condition of the myocardium exists prior to thyrotoxicosis, with organic lesion of the coronaries. This condition is "revealed" by the intervening thyroidal pathology (thyrotoxicosis + ischemic cardiopathy)[1].

In the case described by us of acute myocardial ischemia with thyrotoxicosic crisis, the therapy based on antiarrhythmic drugs (Digitalis, Verapamil, Dihydroquinidine), coronarodilators (nitroderivatives) and Heparin Calcium, followed by the patient for 20 days, had an effect on cardiac rhythm, making atrial fibrillation regress. It did not, though, have any effect in relation to the myocardial ischemia, which remained unmodified, notwithstanding the use of such drugs. (Fig. 1)

On the other hand, once therapy with a beta blocker (Propranolol) and Propylthiouracil was initiated, we were able to witness both resolution of the myocardial ischemia in the course of about 10 days (Fig. 3) and disappearance in a short time of the clinical symptomology.

The rapidity of the regression of electrocardiographic

signs of ischemia and the clinical improvement are to be attributed to the action of the Propranolol, given that Propylthiouracil needs a greater lapse of time to perform its effects.[5]

Therefore, in the course of acute ischemic cardiopathy associated with thyrotoxicosis, we emphasized the need for early use of the beta blocker, which, besides specific antiischemic action, exercises also "antithyroidal" action, both inhibiting adrenergic tone and blocking the conversion of T4 into T3, the thyroidal hormone notably most active[5].

Summary

The authors describe the case of a patient who, in the course of thyrotoxicosis, presented acute myocardial ischemia which was not affected by therapy with nitroderivatives, calcioantagonists and Heparin Calcium, but promptly regressed with the use of Propranolol and Propylthiouracil.

The authors stress the importance of early use of the beta blocker in the course of acute ischemic cardiopathy associated with thyrotoxicosis.

Key words: Acute myocardial ischemia - Thyrotoxicosis - Propranolol.

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